seen in the severe symptoms which accompany summer diarrhœa. Fermentation processes may be of ectogenous or endogenous origin. The former are concerned almost exclusively with cow's milk and may begin within a few hours after the milk has been drawn. The lactic-acid fermentations are due to the breaking-up of the milk sugar, and vary according to the temperature at which the milk is kept. Endogenous fermentation may be a continuation of the ectogenous, and is easily possible, since the infant's stomach is not rich in hydrochloric acid, by which germs introduced with the food are easily destroyed. Fermentation processes associated with a milk diet, both in the stomach and small intestine, lead to acid dyspepsia and diarrhœa, by the destruction of the milk sugar. If the sugar reaches that portion of the intestine where it may be absorbed, the conditions will then be favorable for the decomposition of albumin. Milk which has undergone ectogenous fermentation will cause acute intoxication, with violent local symptoms of irritation, and sometimes collapse, cyanosis, and dyspnœa. One form of endogenous fermentation is the isolated stomach fermentation with acid vomiting, atony, and ectasis of the organ. Other forms are the small-intestine fermentation with its acid diarrhœa, and the large-intestine fermentation with phenomena of mild colitis. Dyspepsia which is due to starch fermentation is very different from the sugar dyspepsias. In the former there is an acid feculent diarrhœa due to the presence of undigested starch in the lower portion of the bowel, and this tends to develop into chronic intestinal catarrh. After a while there may be symptoms resembling cholera, or the upper portion of the intestine or the stomach may be implicated.

PUBLIC HEALTH.

UNDER THE CHARGE OF

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WEIL'S DISEASE.

Physicians in the Southern States, where bilious fevers of every form are rife, may be able to render valuable aid in the solution of the question whether "Weil's Disease" is to be recognized as a distinct specific and communicable disease, or to be considered only as a form of relapsing fever intensified by coexisting or previous malarial poisoning. It appears to be more or less prevalent in Egypt, Syria, and the Balkan States, and a number of accurate and careful observations have, during the past year, been published in the Austrian and Slavonian medical journals. Dr. Frantisek Sumbera[1] reports three cases, Staff-surgeon Th. Hueber[2] four, and Dr. Justyn Karlinski[3] five; the first two contending for its specific character, which is doubted by the last, on the grounds that all his patients had suffered from malaria, that relapsing fever was prevalent in the district, the brother of one being in the hospital for it at the same time, and that he found in the blood of all a spirillus very like that of relapsing fever, though somewhat smaller and more comma-shaped.

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[1] Dr. Frantisek Sumbera: Sbornik Lekarsky, iii. 2, 3, p. 298. 1890.

[2] Dr. Th. Hueber: Deutsch. milit. ärztl. Zeitschr., xix. 1. 1890.

[3] Dr. Justyn Karlinski: Fortsch. d. Med., viii. 5. 1890.

Sumbera's patients, on the other hand, tradesmen between forty and fifty years of age, and Hueber's, soldiers from twenty-two to twenty-five years, had all been in good health up to the date of their attacks.

Sumbera's three cases and one of Hueber's ended fatally, the other three recovering, though very slowly. Only one of Karlinski's died, the other four making rapid recoveries, notwithstanding their previous enfeebled health. It is, therefore, by no means improbable that Karlinski interpreted the nature of their illness correctly, and that he had to deal with quite a different disease from that described by Sumbera and Hueber, in whose cases the course and symptoms of the attack and the post-mortem appearances presented so remarkable a uniformity, that one description will serve for all:

It set in suddenly, with rigors, headache, vomiting, fever, and pains in the back and limbs. Jaundice appeared variously between the second and fifth days. Angina was in Hueber's cases a distressing symptom. The jaundice was soon followed by marked enlargement of the liver, spleen, and inguinal glands. The urine was scanty and high-colored, albuminous or bloody; there was bilious or bloody diarrhœa, the breath was extremely offensive, and the liver and spleen tender. Death occurred in all on the ninth or tenth day, preceded by low delirium and coma. Post-mortem examination in all four fatal cases showed general icterus, fatty degeneration of heart, liver, etc.; hæmorrhagic pachymeningitis; the liver deeply bile-stained, and with dull swelling of the parenchyma; acute interstitial hæmorrhagic nephritis; and internal hæmorrhages in various organs. Neither observer was able to detect the spirillus in the blood.

Wassilief identifies this disease with the typhus biliosus or icterodes kartulis of Smyrna; though he admits that the ill-trained practitioners of Asiatic Turkey and Egypt may mix up many cases of relapsing and pernicious malarial fevers with the true Weil's disease.

ETIOLOGY OF TETANUS.

Tetanus, as distinguished from the tetanic spasms of strychnine-poisoning, and the opisthotonus, etc., incident to some myelites, has generally been considered as a disease, or aggregate of phenomena, consisting essentially of a tonic spasm of all the muscles of the body, beginning with those of the jaws and neck, and ending with those of respiration. Two forms have been recognized—the traumatic and the idiopathic; the former always following the reception of some wound, either one into which dirt has entered, or which has taken on an unhealthy character; the other, in which no such cause has been discovered, has been attributed to exposure to cold and wet—such an explanation being, however, based on negative evidence.

But the researches of Carle and Rattone, Nicolaier, Rosenbach, Briéger Beumer, Langer, and Eiselsberg, leave no room for doubt that it is properly to be classed among specific diseases, and that the phenomena are caused by the entrance into the organism of a microbe which exists outside of the body, and is widely or almost universally distributed in earth and dust. It has been induced in animals by the injection of emulsions of, or the juice expressed from, the sciatic nerve of men or other animals that have died of the disease. Nicolaier used common garden soil with success, though the same earth, when baked or otherwise sterilized, merely set up local irritation and abscesses. Rosenberg discovered and used a bacillus present in the substance of the nerves of tetanic individuals; Briéger employed a chemical product derived from pure cultures of the bacillus; Beumer used all these means; and Eiselsberg has recently reinvestigated the whole question.

The first impulse to research in this direction was given by Drs. Carle and Rattone, who, observing an extraordinary proportion of cases of tetanus among a number of workmen admitted to the hospital in consequence of injuries received by the fall of some old buildings in course of demolition, concluded that there must have been some common and local cause, and thought that it was most probably to be found in the dust that had entered the wounds. Inserting some of the dust gathered from the place into the subcutaneous connective tissue of various animals, they produced tetanus in the larger proportion.

Langer reported the case of five horses who died of tetanus after castration with the same écraseur, which, having been subsequently boiled in oil, was used without ill results.

Eiselsberg, having verified by experiment the results obtained by all previous observers, comes to the conclusion that the belief in the spontaneous origin of some cases of tetanus, like those of erysipelas, is based on errors of observation, some abrasion being present though unnoticed.

The greater frequency of tetanus after wounds, especially lacerated wounds of the hands and feet, is easily accounted for by the fact that, in the acts of grasping or treading, such wounds are most likely to be attended by the entrance of dirt.

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